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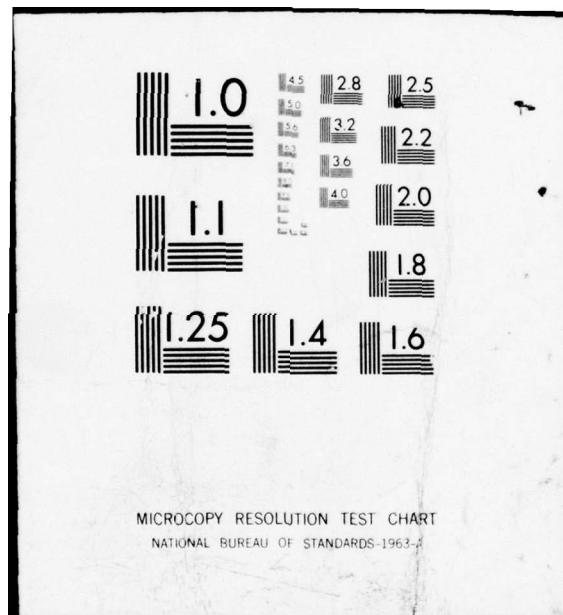
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REPORT NO. T 42/76

**RESUSCITATION OF  
ACCIDENTAL HYPOTHERMIA VICTIMS**

**U S ARMY RESEARCH INSTITUTE  
OF  
ENVIRONMENTAL MEDICINE  
Natick, Massachusetts**

**JUNE 1976**



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**UNITED STATES ARMY  
MEDICAL RESEARCH & DEVELOPMENT COMMAND**

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TECHNICAL REPORT

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RESUSCITATION OF ACCIDENTAL HYPOTHERMIA VICTIMS

by

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Acknowledgements: Dr. William Doolittle, Dr. Robert Boswell,  
Dr. James Chandler, Dr. Mark Cunningham, Dr. James Rabb,  
Dr. Richard Weiskopf, and COL George Smith

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US Army Research Institute of Environmental Medicine  
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ABSTRACT

Accidental hypothermia or whole body cooling is a serious medical emergency. The physician is often faced with a critically ill patient and is often uncertain of how to approach the clinical management and resuscitation of these patients. This paper outlines the physiology of hypothermia and describes the major decision making points in the resuscitation procedure and attempts to define the approach to returning these patients to a normal physiologic state and temperature.

Special reference is given to utilization of internal methods of rewarming, in particular, warm peritoneal dialysis and its effect on hypothermic patients.



INTRODUCTION

This is a composite of the available clinical and experimental literature on clinical and experimental resuscitation of hypothermia victims. It is meant to be a basis for decision making by the physician when hypothermia is suspected. A number of individuals within and outside this Institute have contributed to this document and are cited in the literature review section, this author is merely the current point-of-contact at USARIEM. Discussion, questions, and further contributions are encouraged. The overall aim is to improve medical care and survival statistics for accidental hypothermia victims.

## RESUSCITATION OF ACCIDENTAL HYPOTHERMIA VICTIMS

The recognition of hypothermia as a serious medical emergency is the first step to successful resuscitation. Patients often present cold, cyanotic and pale, stiff as if in rigor with no palpable pulse, no audible heart sounds, no visible respiratory excursions and fixed pupils. They may be in various states of undress and if cooled in a crouched or huddled position impossible to straighten out on an examination table. Their EKG may be extremely bizarre ranging from flat to ventricular fibrillation. Because patients have been successfully resuscitated at core temperatures of 64°F with flat EKGs, the axiom here is "no one is dead until he is warm and dead; attempt rewarming and resuscitation of all cold patients."

There are essentially three types of hypothermia: acute, subacute and chronic, and each has specific requirements for resuscitation and clinical management. Acute hypothermia results from rapid cooling, such as seen in cold water immersion. This acute drop in core temperature is accompanied by few metabolic, electrolyte, and pH abnormalities other than those caused by the direct effect of temperature. The slow cooling rate of chronic hypothermia is usually produced by alcoholic stupor, barbiturate overdose, endocrinopathies, stroke, etc., that subject a person to long-term cold exposure. Slow cooling produces severe alterations in pH, electrolyte balance, and serious alterations in fluid volume. This occurs as the normal physiologic defense mechanisms against cold attempt to counteract the cooling process. The subacute hypothermic patient falls somewhere inbetween, that is, as alcohol

inhibition, rain, heavy winds, poor clothing, etc., subject him to varying cooling rates.

Careful analysis of the immediate prior history can lead to determination of both how and at what rate the patient became hypothermic. Both will impact on how they are handled in the emergency room and in the hospital. Keep in mind that freezing temperatures are not necessary for the production of hypothermic patients.

Resuscitation Tips:

1. Low reading clinical thermometers should be readily available in the emergency room.
2. Core temperature should be taken rectally and is a reliable indication of the progress of rewarming.
3. Careful handling of the patient is essential. Any changes in body position or rough handling can initiate ventricular fibrillation.
4. The blood glucose levels of hypothermic individuals may give a clue to the type of cooling that occurred. Acute hypothermia produces hyperglycemia, while chronic and subacute cooling produces hypoglycemia. The long term shivering of the chronic hypothermic utilizes vast amounts of blood glucose, and, conversion of glycogen to glucose decreases as temperature decreases.
5. Atrial fibrillation is more common in acute than in subacute or chronic hypothermia.

6. Renal failure after rewarming is more common in chronic hypothermia.

7. Current British literature suggests that in acute hypothermia, rapid external rewarming is usually indicated. In chronic hypothermia, they prefer slow rewarming to allow for reversion of metabolic aberrations. This author feels, however, that rapid internal rewarming of chronic hypothermia is a more physiologic procedure.

#### PHYSIOLOGY OF HYPOTHERMIA

Hypothermia, the lowering of core body temperature to 94°F or below is a potentially lethal disorder requiring aggressive therapy. As body temperature decreases below 94°F, central nervous system functions are depressed. Initially, patients exhibit behavioral changes, then depression of consciousness, culminating in coma. The respiratory center is progressively inhibited until apnea supervenes. Cardiac output falls to such extent that despite maximum peripheral resistance the blood pressure falls. The pulse rate decreases. Conduction and heart rhythm abnormalities occur. The "J" wave, various degrees of heart block, atrial premature contractions (APC's), atrial flutter and fibrillation, ventricular premature contractions (VPC's), ventricular tachycardia and fibrillation (VF), and if the patient is cold enough, ventricular standstill can take place. The shift of water out of cells and the intravascular space into the extracellular space as well as decreased renal tubular fluid resorption can render the patient hypovolemic. Some profoundly hypothermic patients exhibit a syndrome similar to



disseminated intravascular coagulation (D.I.C.). Since insulin release and glucose utilization decline with temperature, blood glucose tends to be normal or elevated. Acid-base and electrolyte parameters are little affected by temperature alone but are often deranged by the disorder underlying the hypothermic episode.

The physician must bear in mind that patients who present with hypothermia often have underlying disorders which prevent appropriate physiological responses to the cold environment. Such illnesses include stroke, central nervous system trauma, shock, sedation, use of tranquilizer, or ethanol overdose, endocrinopathies like myxedema and hypoadrenocorticism, hypoglycemia, and old age.

Over medication while cold is a common problem. Subsequent rewarming brings patient into toxic areas for the drugs used. Most drugs are contraindicated in early hypothermia resuscitation.

Much controversy exists over which method of resuscitation, that is, active or passive, external or internal, yields the lowest mortality. The most frequent mechanism of death from hypothermia itself is ventricular fibrillation or standstill. These events can occur at temperatures in the mid 80°sF (27°C) and below. Apnea can occur somewhat higher but usually occurs at lower levels. External warming techniques, active or passive, can actually increase the likelihood of fibrillation during the early phase of resuscitation. The application of heat to the body surface causes peripheral vasodilation, leading to the draining of heat away from core organs, the return of large volumes of cold blood to

the core and thus the lowering of core temperature to increasingly dangerous levels, and a drop in the already low blood pressure. Although this reasoning militates for methods of rewarming the core before the periphery, as through peritoneal dialysis and extracorporeal blood rewarming, the literature suggests that with close monitoring and rapid correction of life-threatening aberrations, external rewarming, both active (with a heated bath or hypothermia blanket), or passive (by wrapping the patient in blankets to prohibit the escape of body heat) yield high survival rates. The author feels that it is physiologically more reasonable to use active than passive methods.

Because of the potential for cardiopulmonary death, the hypothermic patient must be admitted to the intensive care unit. Skull and chest x-ray, blood gases electrocardiogram, blood count, BUN, creatinine, electrolytes, amylase, calcium, blood sugar, fibrinogen, prothrombin time, and platelet count will help in immediate management. If possible the attending physician should be cognizant of the mechanism of the patient's loss of proper thermoregulation. Continuous electrocardiographic monitoring should be instituted. Bizarre EKG tracings are to be expected. Respiratory support including intubation and mechanical ventilation is almost mandatory to keep the supply of oxygen ahead of the rewarming organ demand. Care should be taken during intubation as any rough manipulation can lead to ventricular fibrillation. Ventricular premature contractions are abolished by lidocaine infusion and correction of hypoxia and acidosis. APC's, atrial flutter, and fibrillation will spontaneously revert to normal without medication as cardiac temperature approaches normal. Atropine and electrical pacing have

little beneficial affect on conduction in the hypothermic heart. On the contrary the irritation of the myocardium by the pacemaker electrode itself or by its discharge can lead to VF. If ventricular tachycardia fails to respond to lidocaine or if VF takes place, rapid extracorporeal blood rewarming must be instituted immediately. Because the hypothermic heart is unresponsive to countershock, cardiac temperature must be raised before cardioversion can be successfully accomplished. In such emergencies, the cardiopulmonary bypass machine, equipped with heat exchanger, connected to the femoral artery and vein has been successfully employed. Like any cardiac arrest this situation calls for continuous closed cardiac compression and forced ventilation until the appropriate machines can be placed in operation.

Hypoxia and acidosis are major factors predisposing to ventricular arrhythmias. pH,  $P_{CO_2}$  and  $P_{O_2}$  may appear to be low but may in fact be correct for the organ and brain demand at the depressed temperatures seen in hypothermia. Blood gases and pH, corrected for temperature, should be determined, and abnormalities corrected by adjustment of respiratory parameters or bicarbonate infusion, whichever is necessary but do not overmedicate. Intubation and suction may be necessary to manage bronchorrhea, the physiological response of the airway to exposure to cold air. The rate of spontaneous respiration will increase as the temperature rises.

Maintenance of the central venous pressure at 5-10cm water, with suitable volume expanders, will insure that intravascular fluid volume keeps pace with the capacity of the intravascular space, enlarging in

response to peripheral vasodilation which in turn is caused by external rewarming. Thus, when cardiac temperatures and correspondingly cardiac output and heart rate begin to rise, blood pressure will follow suit. Avoid the use of pressor agents which have no effect on the maximally constricted vessels but which increase the likelihood of ventricular arrhythmias. Similarly, in order to avoid myocardial irritation leading to VF the CVP catheter tip should not be advanced into the heart until some degree of rewarming has occurred and the myocardium is not exceptionally sensitive to physical irritation by the catheter tip. As with respiration, the heart rate will rise spontaneously with temperature. Begin intravenous heparin therapy if clotting tests indicate the occurrence of a DIC-like syndrome.

Other fluid, electrolyte and metabolic abnormalities should be treated as the clinical situation dictates. Therapeutic doses of steroids may be given if hypoadrenocorticism is suspected. Because of the high failure rate of resuscitation of hypothermic myxedematous patients, the latter state must be recognized and treated immediately. After the patient's condition has stabilized, perform whatever additional studies are called for to determine the disease process underlying the hypothermic episode.

#### HYPOTHERMIA CHECKLIST

1. Recognize that the patient is hypothermic - use low temperature thermometer.
2. For patients with compromised mental status or cardiovascular irregularities intensive care is necessary.



3. History of predisposing disease - (Myxedema, hypoadrenocorticism, etc.).
4. Begin, continuous or frequent temperature recording with low temperature recording thermistor or thermometer.
5. Install I.V., (possibly an arterial line), C.V.P., foley catheter (may be extremely difficult).
6. Begin continuous cardiac monitoring.
7. Frequently monitor vital signs and urinary output (at least every hour or more frequently as necessary during rewarming).
8. Wrap patient in rewarming blanket and set to as high a temperature as can be tolerated without burning the patient (104-110°F).
9. Give respiratory support - oxygen by mask or by endotracheal tube (may produce VF) with mechanical ventilation - Aim for high  $PO_2$  normal pH and  $PCO_2$ , and clearance of secretions. Monitor arterial gases and pH as frequently as necessary.
10. Tests: CBC, BUN, creatinine, electrolytes, glucose, amylase, calcium, fibrinogen, prothrombin time, platelet count, chest and skull x-rays, 12 lead E.K.G., arterial blood gases and pH (corrected to core temperature).
11. Maintain C.V.P. between 5 and 10cm with appropriate expanders or fluids calculated to correct electrolyte imbalance gradually.
12. Give bicarbonate to correct acidosis.
13. Treat VPC's with standard boluses 15mg/kg of lidocaine and the correction of hypoxia and acidosis. If ventricular tachycardia, fibrillation, or standstill occur begin closed-chest cardiac compression and assisted

ventilation until extracorporeal blood rewarming can be instituted with cardiopulmonary bypass with heat exchanger. Electrical cardioversion will succeed when the heart warms sufficiently. Atrial premature contractions, flutter and fibrillation will revert to normal with rewarming.

14. Give therapeutic doses of corticosteroids or thyroid hormone if called for.
15. Give heparin for a D.I.C. like syndrome.

THERAPEUTIC OUTLINE FOR THE HYPOTHERMIC PATIENT  
WITH CONSIDERATION OF PERITONEAL DIALYSIS

I. Immediate cardiorespiratory support will, of course, be the first concern, but in the severely hypothermic patient, vigor and rates of resuscitation and dosages of medications should be reduced until patient has begun to reach a more normal temperature.

II.. Respiratory considerations:

- A. Initially respiration is depressed - decreased rate, volume.
- B. Must secure a patent airway which probably means intubation.
- C. Oxygen frequently should be given by mask or endotracheal tube. (tube may produce VF).
- D. Bronchorrhea is common response to cold injury.
- E. Chest x-ray will be required later.

III. EKG and continuous rectal temperature monitoring needed initially to determine if patient is in fact dead.

A. Criteria to proceed:

- 1. Any electrical activity on EKG (may look artifactual).
- 2. Any respiratory effort.
- 3. Absence of pulse, blood pressure, or heart sounds are not adequate for pronouncing death. Neither pupillary dilation nor non-response to light is enough. DTR's may also be absent.

B. Pronouncing dead:

1. May be very difficult decision - probably only guide line is that resuscitation and rewarming of all patients must be attempted.
2. History of long term exposure if A-1,2,3 absent.
3. Rectal temperature less than 50°F.

IV. Rewarming should follow closely with resuscitative efforts.

A. Establish IV line CVP line (cut down may be necessary as venous filling is poor and vessels will be difficult to raise. CVP tip must be kept out of heart early to prevent arrhythmia. Maintain CVP between 5-10 cm water with appropriate fluids, volume expanders. Warm IV fluids to 42°C before use.

B. Concentrate on core rewarming (i.e., peritoneal dialysis) and specifically avoid the application of shell heat initially.

C. Gray zone of when to dialyze.

1. At 90 degrees or greater, one can probably use conservative modalities (i.e., active external rewarming).
2. At 87 degrees or less, dialysis should definitely be utilized.
3. Some recommend dialysis if less than 94°F.

D. Monitor urinary system. Record output, measure specific gravity, Na, and K.

E. Baseline blood studies - probably should not use as indicator for immediate treatment - CBC, Na, K, Cl, CO<sub>2</sub>, BUN, amylase, glucose, pH, PO<sub>2</sub>, PCO<sub>2</sub>, Ca, fibrinogen, platelets, pro time.



F. Dialysis protocol should proceed as per routine in cases of dialysis for renal failure except that exchanges are far more rapid. Bottles should be heated to 45°C and fluid will be about 40-42°C after passage through tubing and arrival at abdomen. Each exchange should take approximately 20 minutes.

Two liters should be run in as fast as bottle elevation and tubing length will allow.

1. Add NO K to dialysate due to possibility of acute renal failure and sensitization of myocardium to VF.
2. Add 1000 units heparin per bottle to prevent clotting of dialysis system.
3. Culture first, fifth, and every tenth exchange.

V. Complications of hypothermia.

A. Cardiac - arrhythmias are biggest concern.

1. At low temperatures may have intractable response in cardioversion and may wind up:
  - a. Trying repeated ineffective shocking causing chest burns.
  - b. Using entirely too much drug for effect at the hypothermic level which then winds up at toxic levels upon rewarming.
2. Xylocaine is probably drug of choice for most arrhythmias given at 15 mg/kg in a bolus or 100 mg total.
3. Defibrillate prn.

4. Atrial premature contractions, flutter, and fibrillation should revert to normal with warming.

5. With only slow normal EKG/ cardiac response, one probably need not apply active CPR

B. Psychiatric problems may result.

1. Patient must be restrained on rewarming due to possible hyperactivity, disorientation, etc.

2. After rewarming, patient may look extremely well but should not be released without adequate observation period. (24 hrs. plus).

C. Pneumonia may ensue. Prophylactic antibiotics should be considered.

1. Poor respiratory effort.

2. Bronchorrhea due to cold injury.

D. Renal failure - be forewarned by hypertension post rewarm.

E. Pancreatitis is a common post rewarming sequella in all types of rewarming.

F. Diabetic ketoacidosis.

G. Disseminated intravascular coagulation - Rx with heparin.

H. Myocardial infarction.

I. Gastrointestinal bleed.

J. Hypotensive episodes.

K. Peritoneal infections secondary to dialysis.

L. Ileus.

Patients should be kept absolutely NPO. Continuous monitoring of rectal temperature, EKG, and blood pressure is absolutely essential.

## SUMMARY

Once diagnosed, start rapidly on rewarming then continuously monitor with minimal interference as the patient recovers.

Further questions can be directed to:

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The opinions and assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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Dr. Mark Cunningham  
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COL George Smith  
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NOTE:

Unpublished Report on Hypothermia presented at UROMED Conference  
5 March 1975, Gol Norway: Malm, Ole J. Accidental Hypothermia.

An excellent review article with 428 references: Little, D.M.  
Hypothermia. Anesthesiology. 842-877, Nov-Dec, 1959.

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